

The Dynamics of EEG Entropy

M. Ignaccolo¹, M. Latka², W. Jernajczyk³, P. Grigolini⁴ and B.J. West^{1,5}

1) Physics Department, Duke University, Durham, NC

2) Institute of Biomedical Engineering, Wrocław University of Technology, Wrocław, Poland

3) Department of Clinical Neurophysiology, Institute of Psychiatry and Neurology, Warsaw, Poland

4) Center for Nonlinear Science, University of North Texas, Denton, TX

5) Mathematics and Information Science Directorate, Army Research Office

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EEG time series are analyzed using the diffusion entropy method. The resulting EEG entropy manifests short-time scaling, asymptotic saturation and an attenuated alpha-rhythm modulation. These properties are faithfully modeled by a phenomenological Langevin equation interpreted within a neural network context.

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I. INTRODUCTION

In the nearly one hundred years since the electroencephalogram (EEG) was introduced into neuroscience there have been a variety of methods used in attempts to establish a taxonomy of EEG patterns in order to delineate the correspondence between brain wave patterns and brain activity. Over that time the single channel EEG time series has been interpreted as consisting of relatively slow regular variations called *signal* and the relatively rapid erratic fluctuations called *noise*. This separation implies that the signal contains information about the EEG channels in the brain, whereas the erratic fluctuations are a property of a channel's environment and does not contain any useful information. Recent studies have refined this engineering model and extracted information from the random fluctuations by concentrating on what is believed to be the scaling behavior of the time series. Analysis of the second moment of the single channel time series shows an algebraic scaling in time, i.e., $\langle X(t)^2 \rangle \propto t^{2H}$. The brackets denote a suitably defined averaging procedure and detrended fluctuation analysis (DFA) [1], which measures the standard deviation of the detrended fluctuations, has been the method of choice for the recent analysis of EEG data [2]. Buiatti et al. [3] use DFA to show that specific task-demands can modify the temporal scale-free dynamics of the ongoing brain activity as measured by the scaling index.

The 'signal' parts of the EEG time series are called waves or rhythms. The nature and scope of these waves have been widely investigated, see Başar [4] for a review. The alpha rhythm (7-12 Hz) has been shown to be typical of awake individuals under no stimulation. Başar, along with colleagues, have developed an integrative theory of alpha oscillations in brain functioning [5]. They hypothesize that there is not one, but several alpha generators distributed within the brain and note that the alpha rhythm may act as a nonlinear clock in the manner suggested by Wiener [6] to serve as a gating function to facilitate the association mechanisms in the brain.

As a measure of order/disorder, entropy has been used to characterize EEG signals. Schlögl et al. [8] measured the entropy of 16 bits EEG polysomnographic records and found it in the range of 8-11 bits. Inouye et al. [9] employed spectral entropy, as defined by the Fourier power spectrum, but the fact that EEG time series are not stationary, in the sense that the autocorrelation function is not simply a function of the two-time difference, obviates the use of Fourier transforms. Subsequently, wavelet entropy was used by Rosso et al. [10] to study the order/disorder dynamics in short duration EEG signals including evoked response potentials. Patel et al. [7], using a combination of MRI and entropy maximization, demonstrated that the generators of alpha rhythm are mainly concentrated over the posterior regions of the cortex.

The diffusion entropy (DE) method [11] has been successfully used to discriminate between the contributions of the low frequency waves (signal) and the high frequency fluctuations (noise), e.g., the influence of the seasons on the daily number of teen births in Texas [12] and the effect of solar cycles on the statistics of solar flares [13]. In this Letter we use the DE method to provide insight on the low/high frequency dynamics of EEG time series.

II. EEG ANALYSIS

Each single channel recording of the EEG time series consists of a sequence of $N + 1$ data points, and the difference between successive data points is denoted by ξ_j for $j = 1, 2, \dots, N$. For the DE analysis a set of stochastic variables

$X_k(t)$ is constructed from the differenced data points in the following way

$$X_k(t) = \sum_{j=k}^{k+t} \xi_j, \quad k = 1, 2, \dots, N - t + 1 \quad (1)$$

to obtain $M = N - t + 1$ replicas of a stochastic trajectory by means of overlapping windows of length t . This ensemble of trajectories, generated by the EEG time series, is used to construct the histogram using the number of trajectories falling in a specified interval to estimate the *pdf* $p(x, t)$. The pdf is then used to calculate the information entropy, a quantity introduced in discrete form for coding information by Shannon [14] and in continuous form for studying the problem of noise and messages in electrical filters by Wiener [15]. We use the latter form here,

$$S(t) = - \int p(x, t) \log_2 p(x, t) dx. \quad (2)$$

The entropy $S(t)$ of Eq. (2) assumes a simple analytical form if the pdf of the diffusion process satisfies the scaling relation:

$$p(x, t) = \frac{1}{\sigma(t)} F\left(\frac{x}{\sigma(t)}\right) \Rightarrow S(t) = C + \log_2 \sigma(t) \quad (3)$$

where $C = - \int F(y) \log_2 F(y) dy$ is a constant and $\sigma(t)$ for a gaussian diffusion process is the time-dependent standard deviation $\sigma(t)$. More generally an α -stable Lévy process also scales in this way, in which case $\sigma(t)$ is more general than the standard deviation of the underlying process.

If the time series were scaling, as assumed in a number of analyses of EEG data [2], the 'variance' would be $\sigma(t) \propto t^\delta$ and the entropy graphed versus time on log-linear graph paper would increase linearly with slope δ . Consequently, the way in which the entropy for a time series scales is indicative of the scaling behavior of the time series. Note that in a simple diffusive process this index is equal to the one obtained from the second moment, that is, $\delta = H$. However, in general, even when there is scaling $\delta \neq H$ and in the case of EEG time series we establish that there is no scaling at all.

We now consider EEG signals of 20 awake individuals in the absence of external stimulations (quiet, closed eyes). EEG signals were recorded using the 10-20 international recording scheme. For 8 individuals only the channels O1, O2, C3 and C4 were recorded, for the remaining 12 individuals all the channels are available. To have a coherent database, we restrict our analysis to the channels O1, O2, C3 and C4, which are the channels traditionally used in sleep studies. The sampling frequency of all EEG records is 250 Hz. Durations of EEG records vary from 55s to 400s with an average duration of 128s.

Fig. 1 shows the DE of the EEG increments for the somnographic channels O1, O2, C3 and C4 of a single individual. We see how for each channel the DE: 1) reaches a saturation level for each channel, 2) has an "alpha" (~ 7.6 Hz in the case of this individual) modulation which is attenuated with time, and 3) has a small amplitude residual asymptotic modulation. The early-time modulation, with variable frequency in the alpha range and variable amplitude, is observed in the somnographic channels for all subjects. The saturation effect is present in all channels for all subjects and it should be pointed out that this saturation is neither a consequence of the finite length of the time series, nor of the finite amplitude of the EEG signal. In fact if the data points were randomly rearranged, thereby destroying any long time correlation in the time series, the EEG entropy does not saturate. Consequently, this saturation effect is due to brain dynamics and is not an artifact of the data processing. Robinson [16] observed this saturation in the calculation of the EEG second moment and interpreted it as being due to dendritic filtering. The inset in Fig. 1 depicts the *pdfs* $p_{\text{sat}}(x)$, after the entropy saturation is attained. These distributions have approximately exponential tails.

III. EEG MODEL

The simplest dynamic model, which includes fluctuations, modulation and dissipation, in short, all the properties displayed in Fig. 1, has the form of a Langevin equation. We assume a dissipative linear dynamic process $X(t)$, i.e., an Ornstein-Uhlenbeck process, with a periodic driver having a random amplitude and frequency and an additive random force $\eta(t)$ which is a delta correlated Gaussian process of strength D :

$$\frac{dX(t)}{dt} = -\lambda X(t) + \eta(t) + \sum_{j=0} A_j \chi[I_{j,s}] \sin[2\pi f_j t] \quad (4)$$

The coefficient $\lambda > 0$ defines a negative feedback, $\chi[I_{j,s}] = 1$ when its argument is in the time interval $I_{j,s} = [jt_s, (j+1)t_s]$ and is zero otherwise, and t_s is the 'stability' time after which a new frequency f_j and a new amplitude A_j are selected.

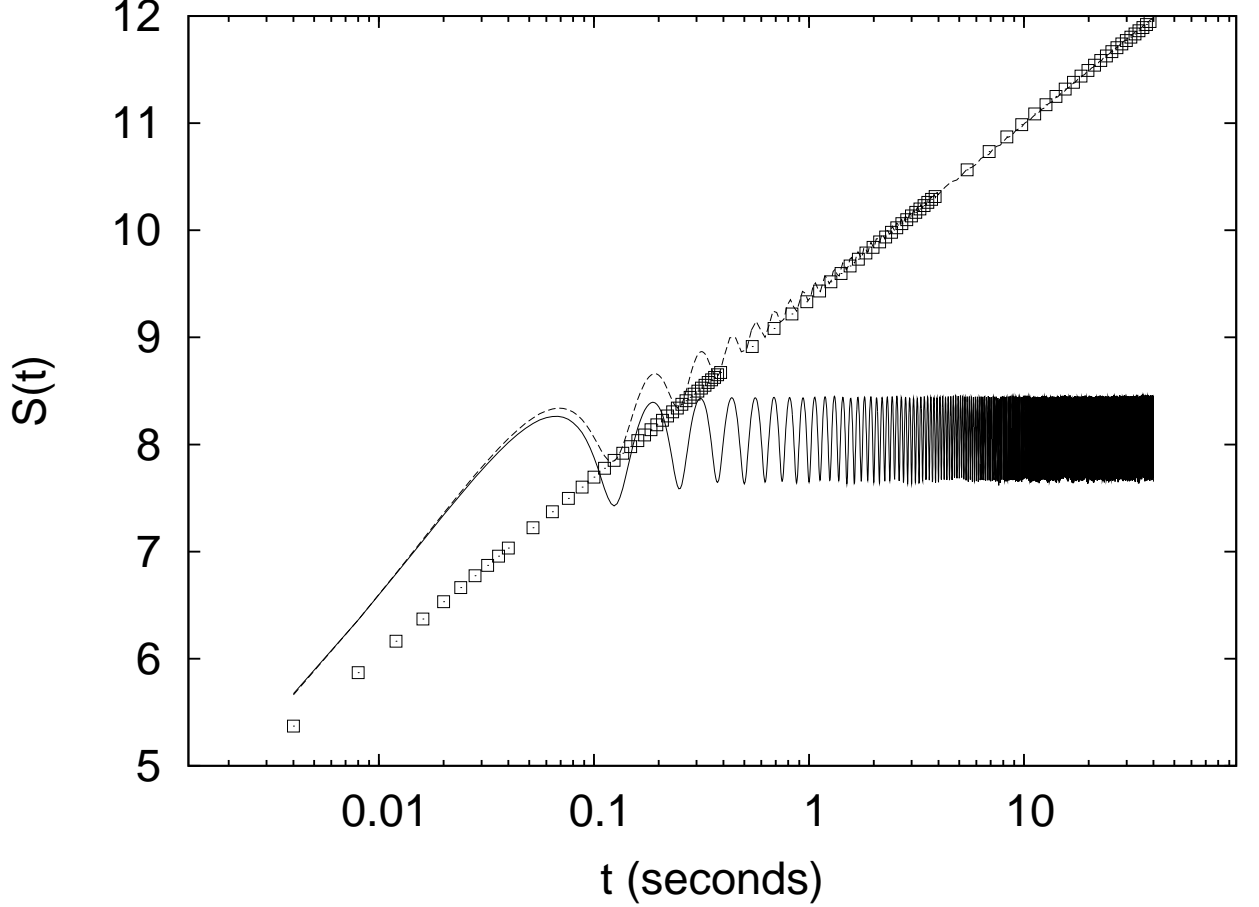


FIG. 1: The diffusion entropy $S(t)$ calculated using the increments of the channels O1, O2, C3 and C4 for one of the 20 subjects considered in this study. The inset depicts the pdfs $p_{\text{sat}}(x)=p(x, t=2000)$ for each channel: squares (O1), circles (O2), upward triangles (C3), and downward triangles (C4).

The values of the frequencies f_j and amplitudes A_j are calculated as follow. First, we calculate the spectral density in the time-frequency domain of time series of EEG increments with a time resolution t_s and a frequency resolution Δf by means of a Windowed Fourier Transform. The spectral density, called the spectrogram (e.g. [17]), is a three-dimensional plot of the spectrum of the EEG increments ξ_j as it changes over time. Then, for each time interval of duration t_s we consider the range of frequencies of the alpha waves, 7-12 Hz, and find which frequency has the maximum amplitude in the spectrogram. This procedure defines the frequency and the amplitude of the time interval considered.

Panel (a) of Fig. 2 shows the spectrogram relative to the increments ξ_j of the channel O1 for the same subject as in Fig. 1. Panels (b) and (c) of Fig. 2 show respectively the sequence of amplitudes A_j (normalized to a maximum amplitude of 1) and of frequencies f_j calculated using the procedure described above. Without an *a priori* knowledge of the typical duration of an alpha wave packet, we set the stability time t_s of Eq. (4) equal to 0.5s. A time resolution of 0.5s and a frequency resolution of $\sim 0.5\text{Hz}$ in the spectrogram represent a reasonable time-frequency localization for our purposes.

Consider the model case where $A_j=0$, for all j , no modulation is present, and Eq. (4) is the Ornstein-Uhlenbeck Langevin equation. In this case the standard deviation of the variable X is $\sigma(t)=\sqrt{D(1-e^{-2\lambda t})/\lambda}$. Consequently, for $t \ll 1/\lambda$ the entropy increases as $S(t)=C'+\frac{1}{2}\log_2 t$, with $C'=C+\log_2 \sqrt{\frac{2D}{\lambda}}$ and a linear-log plot yields a straight line of slope $\delta=0.5$. For $t \gg 1/\lambda$ the entropy reaches the saturation level $S(t)=C+\log_2 \sqrt{\frac{D}{\lambda}}$, yielding an entropy structure similar to that of the EEG data depicted in Fig. 1 without the modulation being present.

When the modulation is present $A_j \neq 0$, Eq. (4) is numerically integrated, and the increments of the dynamic variable X are processed using the DE algorithm. In Fig. 3, we compare the DE obtained via Eq. (4) with that of the channels

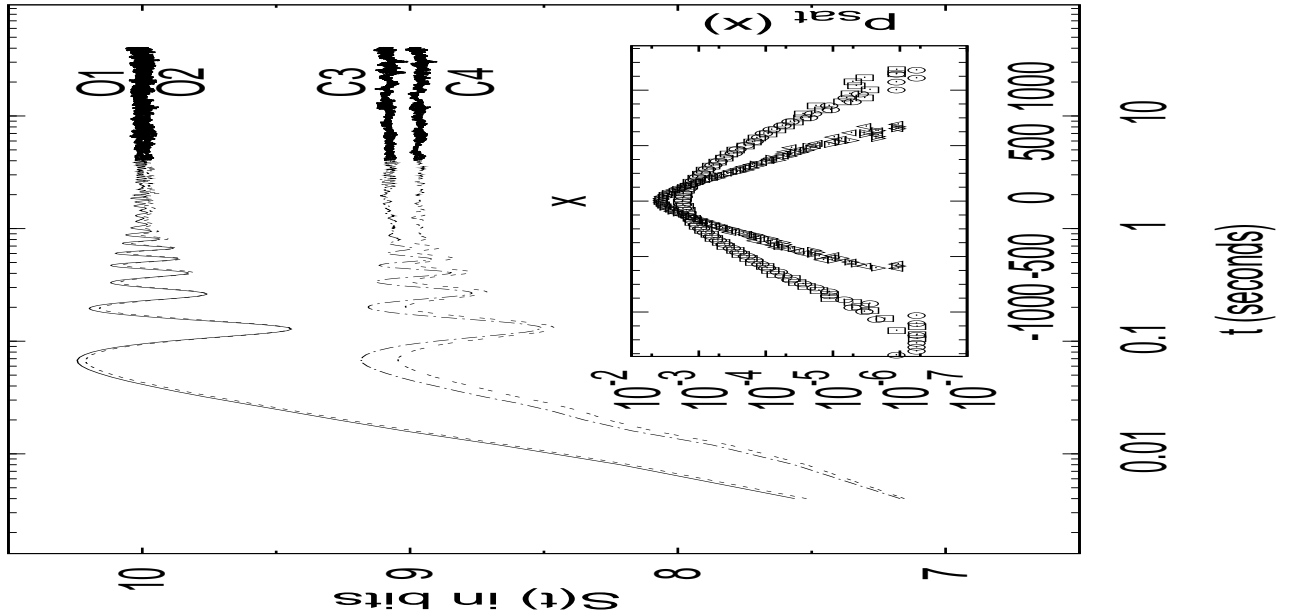


FIG. 2: (a) Spectrogram of the increments of channel O1. We plot the base-10 logarithm of the spectral density. The time resolution is $t_s=0.5$ s, the frequency resolution is $\Delta f=0.5$ Hz. (b) Sequence of the maxima of the spectrogram amplitude (normalized so that the maximum amplitude is 1). This sequence is the sequence of coefficients A_j used in Eq. (4). (c) Sequence of the frequencies corresponding to the amplitude maxima of the spectrogram. This sequence is the sequence of coefficient f_j used in Eq. (4).

O1 and C3, already show in Fig. 1. It is evident that the entropy constructed from the solution to Eq. (4) captures the qualitative and many of the quantitative features of the DE of the EEG increments. Moreover, the asymptotic *pdfs* recorded in the inset also agree with the empirical ones depicted in Fig. 1. In Table 1 we average the phenomenological parameters λ and D for the somnographic channels for the 20 subjects in this study.

TABLE I: The average values (avg.) and the standard deviations (s.d.) of the parameters λ and D of Eq. (4) for all 20 subjects in this study.

EEG channel	λ (avg. \pm s.d.)	D (avg. \pm s.d.)
O1	0.0461 ± 0.0187	16.37 ± 6.88
O2	0.0497 ± 0.0182	16.35 ± 6.72
C3	0.0362 ± 0.0186	10.19 ± 3.90
C4	0.0393 ± 0.0200	10.60 ± 3.72

IV. CONCLUSIONS

The first notable property of the Langevin model is that it reaches a saturation level, indicating that the EEG signal asymptotically carries a maximum amount of information. The EEG entropy does not grow indefinitely as would a random process with long-time correlation. Consequently, the EEG time series do not simply scale as had been previously assumed by a number of investigators [2].

The second notable property of the Langevin model is related to the first and is the dissipation, or negative feedback, produced locally within the channel of interest. The fluctuation-dissipation relation of Einstein is what produces the maximum level of the entropy in a closed physical network, and is given by the ratio of the strength of the additive fluctuations to the dissipation rate. In the more general Langevin equation given here we do not expect the saturation level to be given by this ratio alone, but to depend on the asymptotic value of the 'variance' $\sigma(\infty)$. Note that the

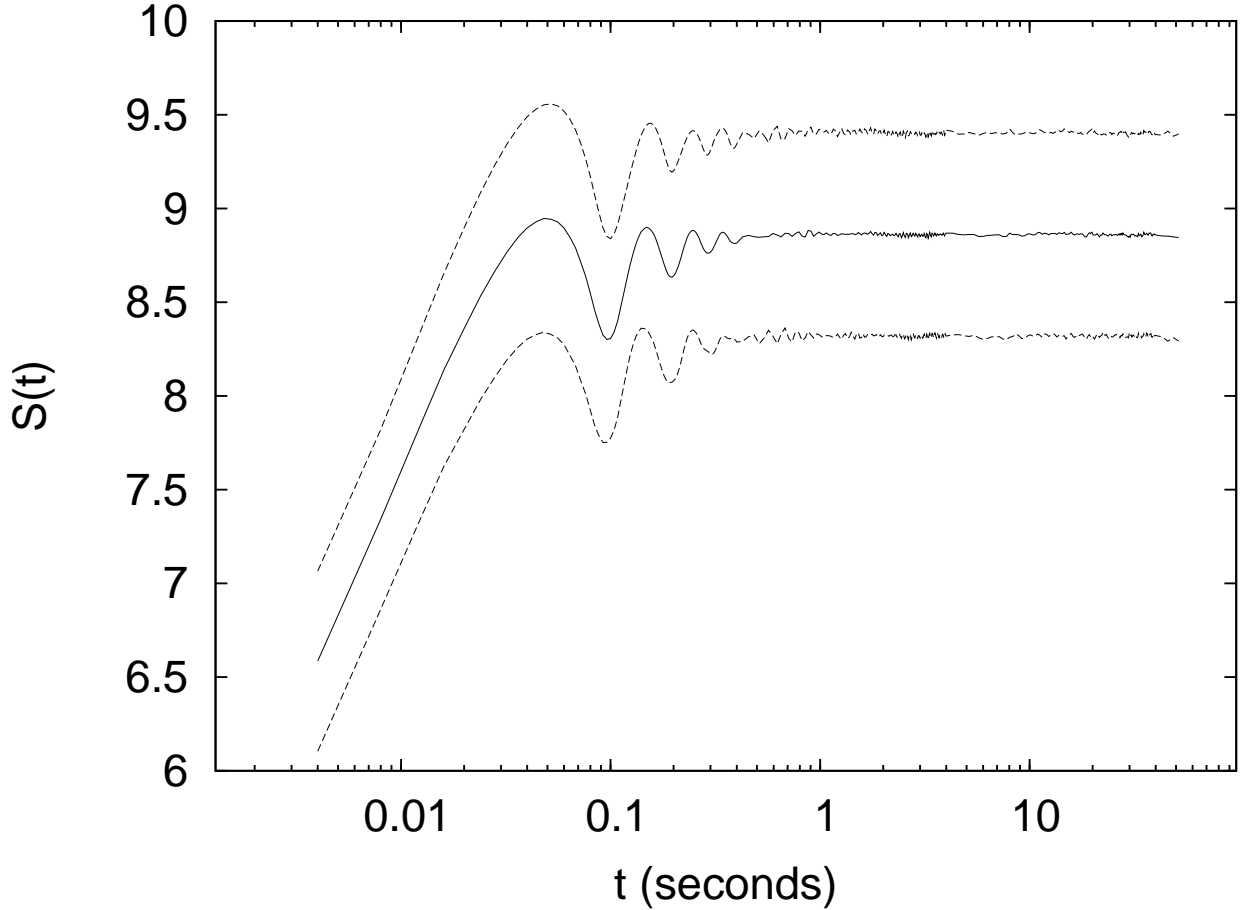


FIG. 3: Comparison between the diffusion entropy of the increments, solid lines, of channel O1 and C3, and diffusion entropy of the increments, points, of the variable X of Eq. (4). The parameters used in Eq. (4) are $\lambda=0.055$ $D=40$ for O1 and $\lambda=0.055$ $D=20$ for C3. Inset show the comparison between the pdfs at saturation $p_{\text{sat}}(X)=p(X, t=2000)$: channels O1 and C3, solid lines, variable X of Eq. (4), squares.

asymptotic 'variance' may not be independent of time, but contains residual information in the form of low amplitude beats because of its dependence on the random near-periodic driver. This mechanism also explains the saturation observed earlier [16] by associating the negative feedback with the dendritic filtering of the signal.

The third notable feature of the Langevin model is the attenuated oscillation of the entropy in time. The attenuation occurs in the EEG entropy because the alpha rhythm is not being generated at one source, but is described as a collective property of the brain in that it is being generated at a number of different locations [5]. Here the influence of the distributed sources is modeled by wave packets that persist for a stability time t_s ; one packet is replaced by another with a slightly different carrier frequency and amplitude chosen from the empirical spectrogram every time interval of length t_s . The concatenation of these wave packets with fluctuating frequencies and amplitudes produces a decoherence that attenuates the modulation of the resulting EEG entropy in time. We show in the sequel that an average over a Gibbs ensemble of phase fluctuations of a harmonic driver in a linear dissipative Langevin equation can be solved analytically to obtain the variance $\sigma(t)^2 = D(1 - e^{-2\lambda t})/\lambda + Ce^{-\gamma t}(1 - \cos \Omega t)$. The constants C and γ are determined by the distribution of amplitudes and frequencies given by the spectrogram, and Ω is the frequency of the oscillation observed in the DE plot. This analytic expression reproduces the results of DE illustrated in Fig. 1. Consequently, this modulation of the EEG entropy indicates that the channel is coupled coherently to the rest of the brain.

The presence of the alpha rhythm modulation masks [12] any early-time scaling property of the EEG dynamics. Eq. (4) is the simplest form of a fluctuation-dissipation process that implies the presence of internal feedback to prevent the occurrence of large excursions of the electric potential inside the brain. The presence of this negative feedback mechanism casts doubts on the possibility of considering an EEG record as the sum of two independent

components, noise and trend, which is the usual assumption made for the DFA method.

Finally, the analysis presented in this Letter support the notion that alpha rhythms [5]: 1) are not generated by one but by a number of spatially distributed sources, whose relative incoherence produces the attenuated modulation of the EEG entropy; 2) are not merely noise but are random and near-periodic; and 3) do not represent passive states, but may determine how different parts of the brain communicate.

Acknowledgments

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